European Journal of Applied Physiology (2025) 125:1349–1359
https://doi.org/10.1007/s00421-024-05687-w

ORIGINAL ARTICLE

Carbohydrate ingestion during prolonged exercise blunts the reduction in power output at the moderate-to-heavy intensity transition

Harrison Dudley-Rode[1] · **Caryn Zinn**[1,2] · **Daniel J. Plews**[1] · **Thanchanok Charoensap**[1] · **Ed Maunder**[1]

Received: 3 September 2024 / Accepted: 4 December 2024 / Published online: 22 December 2024

Abstract

Purpose To determine the effect of carbohydrate ingestion during prolonged exercise on durability of the moderate-to-heavy-intensity transition and severe-intensity performance.

Methods Twelve trained cyclists and triathletes (10 males, 2 females; $\dot{V}O_2$peak, 59 ± 5 mL kg^{-1} min^{-1}; training volume, 14 ± 5 h $week^{-1}$) performed an incremental test and 5-min time trial (TT) without prior exercise (PRE), and after 150 min of moderate-intensity cycling, with ($POST_{CHO}$) and without ($POST_{CON}$) carbohydrate ingestion.

Results Power output at the first ventilatory threshold (VT_1) was lower in $POST_{CHO}$ (225 ± 36 W, Δ -3 ± 2%, $P = 0.027$, $n = 11$) and $POST_{CON}$ (216 ± 35 W, Δ -6 ± 4%, $P = 0.001$, $n = 12$) than PRE (229 ± 37 W, $n = 12$), and lower in $POST_{CON}$ than $POST_{CHO}$ (Δ -7 ± 9 W, Δ -3 ± 4%, $P = 0.019$). Mean power output in the 5-min TT was lower in $POST_{CHO}$ (351 ± 53 W, Δ -4 ± 3%, $P = 0.025$) and $POST_{CON}$ (328 ± 63 W, Δ -10 ± 10%, $P = 0.027$) than PRE (363 ± 55 W), but $POST_{CHO}$ and $POST_{CON}$ were not significantly different (Δ 25 ± 37 W, Δ 9 ± 13%, $P = 0.186$). Blood glucose concentration was maintained in $POST_{CHO}$, and was significantly lower at the 120 and 150-min timepoint in $POST_{CON}$ ($P < 0.05$).

Conclusion These data suggest that durability of the moderate-to-heavy-intensity transition is improved with carbohydrate ingestion. This has implications for training programming and load monitoring.

Keywords Durability · Nutrition · Carbohydrate · Exercise

Abbreviations

CHO	Carbohydrate
VO_2	Rate of oxygen consumption
VO_2peak	Peak rate of oxygen consumption
$\dot{V}_E\,\dot{V}O_2^{-1}$	Ventilatory equivalent for oxygen
VT_1	First ventilatory threshold
VT_2	Second ventilatory threshold
EE	Energy expenditure
PFO	Peak fat oxidation rate

Introduction

The physiological response to exercise can be categorised into three distinct domains (Jones et al. 2019). During exercise in the moderate-intensity domain, there is minimal disturbance to muscle metabolic and ionic homeostasis, and blood lactate concentrations remain close to baseline values. During heavy-intensity exercise, a delayed steady state in blood lactate concentrations, whole-body oxygen consumption ($\dot{V}O_2$), and muscle metabolic and ionic homeostasis (e.g. PCr, Pi, H^+) is achieved, whereas during severe-intensity exercise, no steady state is achieved, and these values progress to a peak or nadir at task failure (Black et al. 2017; Burnley et al. 2010; Jones et al. 2008). Power output at the intensity domain transitions decreases during prolonged exercise, with large between-athletes variability (Clark et al. 2018; Clark et al. 2019a, b; Clark et al. 2019a, b; Gallo et al. 2024; Hamilton et al. 2024; Stevenson et al. 2022). Resilience to the effects of prolonged exercise on the intensity domain transitions has been termed 'durability' (Maunder

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Communicated by Michael I Lindinger.

✉ Ed Maunder
ed.maunder@aut.ac.nz

[1] Sports Performance Research Institute New Zealand, Auckland University of Technology, Auckland, New Zealand

[2] Human Potential Centre, Auckland University of Technology, Auckland, New Zealand

et al. 2021). Therefore, it has been suggested that durability should be considered a key performance determinant (Jones 2023; Maunder et al. 2021).

The reduction in power output at the intensity domain transitions following prolonged exercise could be related to decreased carbohydrate substrate availability (Clark et al. 2019a, b; Clark et al. 2019a, b; Stevenson et al. 2022). Muscle glycogen progressively declines during exercise (Bergström and Hultman 1966, 1967; Hermansen et al. 1967; Karlsson et al. 1974), and depletion of the inter- and intramyofibrillar glycogen stores has been linked with function of the sarcoplasmic reticulum Ca^{2+} ATPase and Na^+, K^+ ATPase (Nielsen et al. 2009, 2011, , 2014, 2022; Ørtenblad et al. 2013). Muscle glycogen depletion renders individual fibers inexcitable (Cairns and Renaud 2023). As the most-oxidative fibres are preferentially recruited during prolonged moderate and heavy exercise, the glycogen stores of type I fibers are depleted fastest (Nielsen et al. 2024). Consequently, the active, excitable fibre pool becomes progressively less oxidative during prolonged exercise, and power production increasingly requires activation of type II fibres, which have lower mitochondrial protein content (Nielsen et al. 2024). Similarly, liver glycogen is depleted during prolonged exercise (Casey et al. 2000; Gonzalez et al. 2015; Stevenson et al. 2009), and liver glucose output provides an additional glucose source for contracting muscles and prevents hypoglycemia (Gonzalez et al. 2016). Preventing hypoglycemia is crucial for avoiding neuroprotective downregulation of motor unit recruitment (Elghobashy et al. 2024; Glace et al. 2019; Nybo 2003). Therefore, muscle and/or liver glycogen depletion may partially explain the reduction in power output achieved at intensity domain transitions during prolonged exercise.

In line with this hypothesis, carbohydrate ingestion mitigates the reduction in power output at the heavy-to-severe-intensity transition during exercise (Clark et al. 2019a, b; Clark et al. 2019a, b). This could plausibly be attributed to muscle glycogen-sparing (Tsintzas et al. 1995, 1996), although many studies have not reported a muscle glycogen-sparing effect of carbohydrate ingestion during prolonged cycling (Coyle et al. 1986; Gonzalez et al. 2015; Jeukendrup et al. 1999). However, the effect of carbohydrate ingestion during prolonged exercise on subcellular glycogen stores has not been assessed. In contrast, carbohydrate ingestion during exercise has been consistently shown to reduce liver glucose output, preserve liver glycogen stores, and prevent hypoglycemia (Bosch et al. 1994; Gonzalez et al. 2015; Hargreaves et al. 1995; Jeukendrup et al. 1999).

The effect of carbohydrate ingestion during prolonged moderate-intensity exercise on durability of the moderate-to-heavy-intensity transition has not been studied. Strategies to improve durability of the moderate-to-heavy-intensity transition have implications for stochastic-intensity events such as road cycling, where performance outcomes are often determined by the ability to produce high work outputs following multiple hours of exercise (Fernández-García et al. 2000; Sanders et al. 2019). More durable athletes are likely to spend less time in the more-demanding heavy- or severe-intensity domains during prolonged exercise prior to a subsequent severe-intensity effort, which may accelerate glycogen depletion and the disturbance of muscle metabolic homeostasis (Black et al. 2017). Indeed, we recently found a strong relationship between durability of the moderate-to-heavy-intensity transition and severe-intensity performance (Hamilton et al. 2024), and others reported that time spent above the moderate-to-heavy-intensity transition during six hours of simulated road cycling was moderately related to loss of severe-intensity time-trial performance (Klaris et al. 2024).

Accordingly, the purpose of this study was to assess the effect of carbohydrate ingestion during prolonged exercise on durability of the moderate-to-heavy-intensity transition and severe-intensity time-trial performance. We hypothesised that carbohydrate ingestion during exercise would partially mitigate the reduction in power output at the moderate-to-heavy-intensity transition, and during a 5-min time trial, following prolonged exercise.

Methods

Participants

Twelve trained cyclists and triathletes were recruited to take part in this investigation (ten males, two females; age, 31 ± 6 years; height, 179.9 ± 5.5 cm; mass, 75.6 ± 6.4 kg; $\dot{V}O_2$peak, 59.3 ± 5.2 mL kg^{-1} min^{-1}; training volume, 14 ± 5.4 h $week^{-1}$; habitual energy intake, 2502 ± 608 kcal day^{-1}) (Table 1). The participants ranged from 20 to 55 years old, were free of illness and musculoskeletal injury (> 3 months), had never had cardiovascular

Table 1 Descriptive participant characteristics

	Male (*n* = 10)	Female (*n* = 2)
Age (years)	34	37
Height (cm)	181.9 ± 3.2	170.0 ± 2.3
Mass (kg)	76.9 ± 5.2	69 ± 9.8
VO_2peak (mL.kg^{-1}.min^{-1})	60.7 ± 4.2	52 ± 5.1
Training volume (h.$week^{-1}$)	14.3 ± 5.7	14.0 ± 5.7
Habitual energy intake (kcal)	2636 ± 494	1899 ± 920
Carbohydrate (%)	47 ± 10	22 ± 3
Fat (%)	34 ± 9	46 ± 12
Protein (%)	19 ± 3	33 ± 15

disease, self-reported training > 8 h·week^{-1}, and had a peak oxygen uptake ($\dot{V}O_2$peak) > 55 mL·kg^{-1}·min^{-1} for males and > 48 mL·kg^{-1}·min^{-1} for females (determined in the first visit) and self-reported best-effort 20-min power output of > 3.5 W·kg^{-1}. A priori sample size estimation indicated a total of 12 participants were required to detect a large magnitude (d = 0.8) effect of carbohydrate ingestion on durability of the moderate-to-heavy-intensity transition with 80% power and a type I error rate of 0.05, using a one-tailed test. A large effect size was used as previous research on the effect of carbohydrate ingestion during exercise on durability of the heavy-to-severe-intensity transition reported a large effect (Clark et al. 2019a, b; Clark et al. 2019a, b). All participants completed a general health screening and provided written informed consent. This study was performed in accordance with the standards of the Declaration of Helsinki, 2013, and the Auckland University of Technology Ethics Committee approved all procedures (23/197).

Study design

This study involved four laboratory visits, adopting a randomized, Latin Square counterbalanced, double-blind, cross-over design, with one characterization trial and three experimental trials (Fig. 1). The first visit was used to assess eligibility, to provide an initial measure of power output at VT_1 and peak oxygen uptake ($\dot{V}O_2$peak), to measure the peak fat oxidation (PFO) rate and gross cycling efficiency, and to familiarize the participants with the 5-min time trial used in subsequent trials. Visits 2–4 were the experimental trials, and took place in a randomized, counterbalanced order. Each experimental trial began in the morning following the consumption of a breakfast containing ~ 1 g·kg^{-1} of carbohydrate and ~ 800 mL of water, and involved measurement of VT_1 using a five-step incremental test and 5-min time-trial performance (i) without prior cycling (PRE), (ii) following 150 min of cycling at 90% of the initial estimate of VT_1, with consumption of carbohydrate at 60 g·h^{-1} ($POST_{CHO}$), and (iii) following 150 min of cycling at 90% of the initial estimate of VT_1, without carbohydrate ($POST_{CON}$). In $POST_{CON}$, participants consumed a calorie-free, taste-matched placebo drink. Participants and researchers were blinded to the condition ($POST_{CHO}$ vs. $POST_{CON}$). Power output at VT_1 was used as an estimate of the moderate-to-heavy-intensity transition (Jamnick et al. 2020), in line with previous work (Gallo et al. 2024; Hamilton et al. 2024; Stevenson et al. 2022). We previously found that VT_1 and the lactate threshold, calculated using the LoglogLT method (Jamnick et al. 2018) produced similar results and responded to prolonged exercise in the same fashion as VT_1 (Stevenson et al. 2022), which supports use of this approach for estimating the moderate-to-heavy-intensity transition in this context.

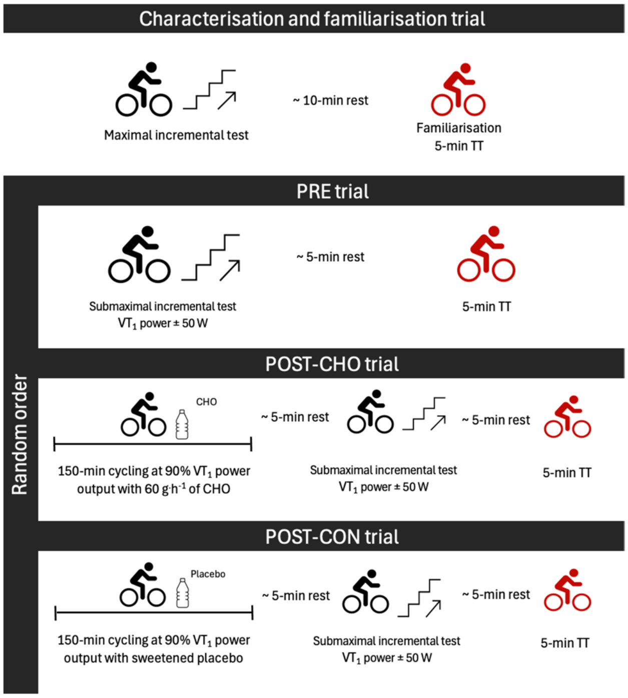

Fig. 1 Schematic overview of the study design. TT, time trial; VT_1, first ventilatory threshold; CHO, carbohydrate

Characterisation trial

Maximal incremental test and familiarisation time trial

Participants arrived in the laboratory having fasted overnight for ~ 10 h, for accurate quantification of substrate utilisation, and having ingested ~ 1–2 L of water in the morning before arrival. Before beginning the session, the height and the body mass of participants were measured. Cycling then commenced on personal road bicycles mounted to a direct-drive smart indoor trainer (Kickr, Wahoo Fitness, GA, USA) at 95 W, with the work rate increased by 35 W every 3 min. Expired gases and heart rate were collected continuously during exercise (TrueOne 2400, ParvoMedics, UT, USA; Polar H10, Polar Electro Oy, Kempele, Finland). Upon confirmation of the second ventilatory threshold (VT_2) via a clear rise in $\dot{V}_E \dot{V}CO_2^{-1}$ and fall in $PetCO_2$, the work rate was increased by 35 W every minute until volitional exhaustion. The $\dot{V}O_2$peak was accepted as the highest 15-s average $\dot{V}O_2$, and VT_1 was determined by identifying the $\dot{V}O_2$ at the first breakpoint in the $\dot{V}O_2$ vs. $\dot{V}_E \cdot \dot{V}O_2^{-1}$ relationship. This $\dot{V}O_2$ was subsequently converted into a power output via simple linear regression of $\dot{V}O_2$ vs. power output during the 3-min stages, using the average $\dot{V}O_2$ in the last minute of each stage. The last minute of expired gas data in each 3-min stage was used to quantify whole-body fat oxidation using standard equations (Eq. 1, 2, Jeukendrup & Wallis 2005).

$$\text{Whole} - \text{body fat oxidation rate } (\text{g} \cdot \text{min}^{-1}) = 1.695 \times \dot{V}O_2 - 1.701 \times \dot{V}CO_2 \tag{1}$$

where $\dot{V}O_2$ and $\dot{V}CO_2$ are in L.min^{-1}

$$\text{Whole} - \text{body rate of energy expenditure } (\text{kcal} \cdot \text{min}^{-1}) = 0.550 \times \dot{V}CO_2 + 4.471 \times \dot{V}O_2 \tag{2}$$

where $\dot{V}O_2$ and $\dot{V}CO_2$ are in L.min-1.

The highest observed rate of whole-body fat oxidation was identified as PFO (Maunder et al. 2022). After completion of the incremental test, participants rested for ~ 10-min prior to completing a 5-min performance time trial. Participants were instructed that the time trial was to be performed with maximum effort, with the goal of achieving the highest possible average power output. Participants were blinded to power output, heart rate, and cadence, but able to see elapsed time. Expired gases were collected during the time trial, with the highest 30-s average $\dot{V}O_2$ accepted as the time-trial $\dot{V}O_2$peak. Following the time trial, participants were instructed on how to accurately record exercise for the week prior and dietary intake for the day prior to the second trial using a smartphone application (Easy Diet Diary Connect, Xyris Software, QLD, Australia). Habitual dietary macronutrient intake was subsequently quantified (Easy Diet Diary, Xyris Software, QLD, Australia). Participants were asked to replicate their exercise and food intake in advance of the third and fourth trial.

Experimental trials

PRE, POST-CHO, and POST-CON assessments of the moderate-to-heavy intensity transition and severe-intensity time-trial performance

Participants returned to the laboratory 5–14 days later to complete the first of the three experimental trials. Participants arrived having consumed a breakfast of their choosing containing ~ 1 g·kg^{-1} of carbohydrate, which was replicated for all experimental trials, and ~ 800 mL of water one hour beforehand. Participants were fitted with a heart rate monitor such that heart rate was recorded continuously throughout the trial. All trials began with a 5-min warm-up at 100 W. Following the warm-up, participants cycled for 150 min at 90% of the VT_1 power output estimated in the characterisation trial in the $POST_{CHO}$ and $POST_{CON}$ trials, but not the PRE trial, with expired gases collected for 4 min every 15 min. Expired gas data were used to quantify rates of whole-body energy expenditure, carbohydrate oxidation, and fat oxidation during the 150-min preload. Heart rate data were separated into 15-min bins during the 150-min preload. In the $POST_{CHO}$ and $POST_{CON}$ trials, participants consumed 250 mL of water every 20 min during the first 120-min of the 150-min preload. In $POST_{CHO}$, water was mixed with a calorie-free electrolyte mix (Electrolytes, Musashi, Vitaco Health, Auckland, New Zealand) and maltodextrin (Pure Maltodextrin, Reactiv, Auckland, New Zealand) such that carbohydrate was consumed at a rate of 60 g·h^{-1} during the first 120-min of the 150-min preload. In $POST_{CON}$, water was mixed with the same calorie-free electrolyte mix with added sweetener (Sugar Fix'd Natural Sweetener, Natvia, VIC, Australia) to match the taste of the $POST_{CHO}$ beverage but be devoid of carbohydrate. Additionally, a fingertip capillary blood glucose measurement was obtained using a blood glucose analyser (Freestyle Optimum Neo, Abbott, Berkshire, United Kingdom) at rest and every 30 min during the 150-min preload in $POST_{CHO}$ and $POST_{CON}$. Participants and researchers were blinded to trial allocation.

Subsequently, the moderate-to-heavy intensity transition was estimated using a five-step incremental test, with continuous collection of expired gases. The first step was 50 W below the VT_1 power output measured in the first laboratory visit, and the power output was increased by 25 W every 4-min, such that the fifth and final step was 50 W above the VT_1 power output measured in the first laboratory visit. The moderate-to-heavy intensity transition power output was measured using the methods previously described for determining VT_1 in the first laboratory visit, but with greater precision given the denser cluster of datapoints around the transition. Following the five-step incremental test, participants cycled at 100 W for 5 min before completing a 5-min performance time trial according to the procedures described above.

Mathematically, loss of power output at VT_1 can be attributed to: (i) reduced mechanical power output at a given rate of metabolic energy expenditure (reduced energetic efficiency) and (ii) a reduction in the rate of metabolic energy expenditure at VT_1 (reduced metabolic power). To determine the contributions made by changes in energetic efficiency and metabolic power to changes in power output at VT_1 in $POST_{CON}$ and $POST_{CHO}$, the rates of metabolic energy expenditure associated with the power output identified at VT_1 in $POST_{CON}$ and $POST_{CHO}$ were calculated. First, the rate of metabolic energy expenditure at each power output during the incremental test in each trial was calculated using the last minute of expired gas data in each 4-min stage (Eq. 2). Subsequently, the relationship between power output and metabolic energy expenditure in each trial was quantified using linear regression, which was then used to estimate the rate of metabolic energy expenditure at the power output VT_1 in each trial. Next, the rate of metabolic energy expenditure associated with VT_1 in $POST_{CON}$ and $POST_{CHO}$ was converted to power output using linear regression of the power output vs. energy expenditure relationship in the *PRE* trial (denoted $POST\text{-}CON_{EE}PRE_{Eff}$, $POST\text{-}CHO_{EE}PRE_{Eff}$). Therefore, $POST\text{-}CON_{EE}PRE_{Eff}$ and $POST\text{-}CHO_{EE}PRE_{Eff}$ identify the theoretical power output that the rate of metabolic energy expenditure measured at VT_1 in $POST_{CON}$ and $POST_{CHO}$ would have produced with the same level of energetic efficiency as in the PRE trial. Accordingly, the contributions to changes in power output at VT_1 made by changes in energetic efficiency and metabolic power, in $POST_{CON}$ and $POST_{CHO}$, were calculated (Eq. 3).

(Eq. 3) Contribution of change in energetic efficiency to change in power output at VT_1 in $POST_{CON}$ and $POST_{CHO} = POST_{CON} - POST\text{-}CON_{EE}PRE_{Eff}, = POST_{CHO} - POST\text{-}CHO_{EE}PRE_{Eff}$.

Contribution of change in metabolic power to change in power output at VT_1 in $POST_{CON}$ and $POST_{CHO} = POST\text{-}CON_{EE}PRE_{Eff} - PRE$, $POST\text{-}CHO_{EE}PRE_{Eff} - PRE$.

where $POST\text{-}CON_{EE}PRE_{Eff}$, $POST\text{-}CHO_{EE}PRE_{Eff}$ is the theoretical power output produced with the metabolic energy expenditure at VT_1 in $POST_{CON}$ and $POST_{CHO}$, respectively, with the gross cycling efficiency in PRE.

Statistical analyses

Data were expressed as mean ± standard deviation and analyzed using GraphPad Prism Version 9.3.1. Statistical significance is inferred when $P \leq 0.05$. The normality of datasets was assessed using the Shapiro–Wilk test. Power output at VT_1 and 5-min time-trial performance were compared between-trials using mixed-effects analyses due to missing data (one participant did not complete $POST_{CHO}$ due to illness), with trial as fixed effects and participant as random effects. Variance was located post hoc using Bonferroni-corrected paired *t*-tests. The $\dot{V}O_2$peak was compared between-trials using a one-way repeated measures ANOVA. Percentage changes were calculated to determine the practical magnitude of differences. Contributions to changes in power output at VT_1 by changes in energetic efficiency and metabolic power were compared between-trials using paired *t*-tests. Whole-body rates of energy expenditure, carbohydrate oxidation, fat oxidation, gross efficiency, blood glucose concentration, and heart rate during the 150-min preload in $POST_{CON}$ and $POST_{CHO}$ were compared using two-way repeated measures analyses of variance, with trial and time as factors. Bonferroni-corrected *t*-tests were used to locate variance. Relationships between habitual dietary macronutrient intake and PFO and durability of power output at VT_1, 5-min time-trial performance, and $\dot{V}O_2$peak were assessed using Pearson's or Spearman's rank-order correlation coefficients (depending on normality) and expressed with 95% confidence intervals.

Results

Characterisation trial

The $\dot{V}O_2$peak measured in the characterisation trial was $4.48 \pm 0.55\ L\cdot min^{-1}$. The estimated power output at VT_1 was 226 ± 38 W, and therefore a power output of 215 ± 36 W was used during the preload phase of the prolonged experimental trials. The estimated power output at VT_2 was 290 ± 38 W and peak fat oxidation was $0.63 \pm 0.16\ g\cdot min^{-1}$.

Prolonged phase

One participant did not complete the $POST_{CHO}$ trial. Hence, the data presented below is $n = 12$ for $POST_{CON}$ and $n = 11$ for $POST_{CHO}$. During the prolonged phase, there was an effect of time for blood glucose concentration ($P = 0.0288$, Fig. 2E) and heart rate ($P < 0.0001$, Fig. 2F). No other effects of time were observed during the prolonged phase. There was an effect of trial for blood glucose concentration ($P = 0.0054$), and a trial-by-time interaction for carbohydrate oxidation ($P = 0.0179$). No other effects of trial or trial-by-time interactions were observed during the prolonged phase. Carbohydrate oxidation rate was significantly greater in $POST_{CHO}$ than $POST_{CON}$ at 150-min ($P = 0.043$). Blood glucose concentration was significantly greater in $POST_{CHO}$ than $POST_{CON}$ at 120- and at 150-min (Fig. 2E).

Effects of prolonged exercise and carbohydrate ingestion

There was a significant effect of trial on power output at VT_1 ($P = 0.0163$). Power output at VT_1 was significantly lower in $POST_{CHO}$ (225 ± 36 W, Δ -7 ± 3 W, Δ -3 ± 2%, $P = 0.0265$) and $POST_{CON}$ (216 ± 35 W vs. 229 ± 37 W, Δ -13 ± 9 W, Δ -6 ± 4%, $P = 0.0011$) than PRE (229 ± 37 W, Fig. 3A). Power output at VT_1 was significantly greater in $POST_{CHO}$ than $POST_{CON}$ (225 ± 36 W vs. 216 ± 35 W, Δ 7 ± 9 W, Δ 3 ± 4%, $P = 0.0186$). The contribution to loss of power output at VT_1 following prolonged exercise made by reduced metabolic power was significantly greater in $POST_{CON}$ than $POST_{CHO}$ ($P = 0.0101$, Fig. 3B). This difference was not significant for loss of energetic efficiency ($P = 0.3131$).

There was a significant effect of trial on 5-min time-trial performance ($P = 0.0266$). Mean power output in the 5-min time trial was significantly lower in $POST_{CHO}$ (351 ± 53 W, Δ -15 ± 12 W, Δ -4 ± 3%, $P = 0.0251$) and $POST_{CON}$ (328 ± 63 W, Δ -35 ± 38 W, Δ -10 ± 10%, $P = 0.0272$) than PRE (363 ± 55 W) (Fig. 4A). Mean power output in the 5-min time trial was

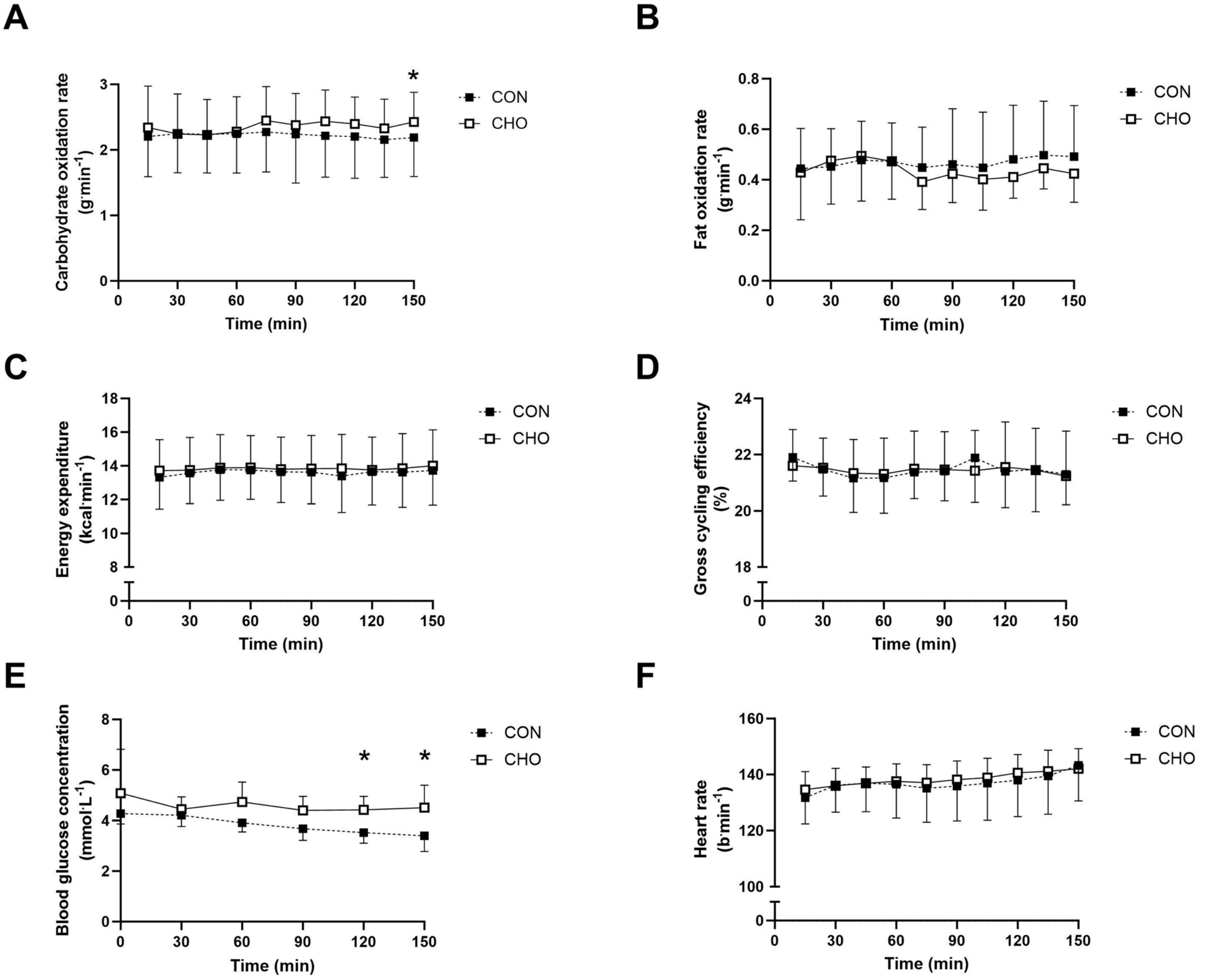

Fig. 2 **A** Carbohydrate oxidation rate, **B** Fat oxidation rate, **C** Energy expenditure, **D** Gross cycling efficiency, **E** Blood glucose concentration, **F** Heart rate during the preload phase of $POST_{CHO}$ and $POST_{CON}$. * denotes $P \leq 0.05$

not significantly different between $POST_{CHO}$ and $POST_{CON}$ (Δ 25 ± 37 W, Δ 9 ± 13%, $P = 0.1859$). There was no significant effect of trial on VO_2peak measured during the 5-min time trial ($P = 0.0831$) (Fig. 4B).

Discussion

The primary aim of this study was to determine the effect of carbohydrate ingestion during prolonged moderate-intensity cycling on durability of the moderate-to-heavy intensity transition and severe-intensity time-trial performance. We hypothesised that carbohydrate ingestion during prolonged cycling would partially mitigate the decline in power output at the moderate-to-heavy intensity transition and 5-min time-trial performance. Our primary observations were that: (i) carbohydrate ingestion during prolonged cycling partially mitigated the decline in power output at the moderate-to-heavy intensity transition and (ii) carbohydrate ingestion did not significantly influence the decline in severe-intensity time-trial performance following prolonged exercise. These data therefore suggest that durability of the moderate-to-heavy intensity transition is related to carbohydrate availability.

In line with our hypothesis, carbohydrate ingestion during prolonged exercise in $POST_{CHO}$ partially mitigated the decline in power output at the moderate-to-heavy intensity transition seen in $POST_{CON}$ (Fig. 3A). Specifically, power output at the moderate-to-heavy intensity transition was ~ 6% lower in $POST_{CON}$ vs. PRE, but this difference was only ~ 3% in $POST_{CHO}$. The reduction in power output at the moderate-to-heavy intensity transition following prolonged exercise aligns with previous research concerned with both the moderate-to-heavy (Hamilton et al. 2024; Gallo et al.

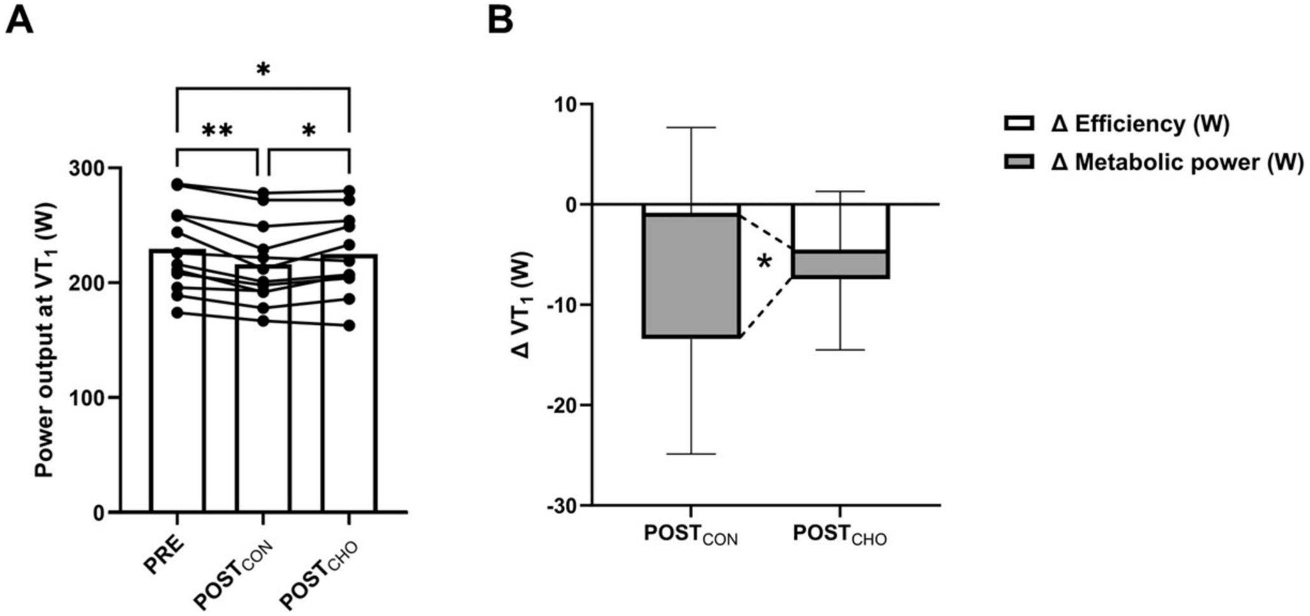

Fig. 3 **A** Power output at the first ventilatory threshold (VT_1) in the PRE, $POST_{CON}$, and $POST_{CHO}$ trials, **B** Contribution to changes in the power output at the first ventilatory threshold (VT_1) by losses in energetic efficiency and metabolic energy expenditure at the threshold after a period of prolonged exercise in the $POST_{CON}$ and $POST_{CHO}$ trials. * denotes $P \leq 0.05$, ** denotes $P \leq 0.01$

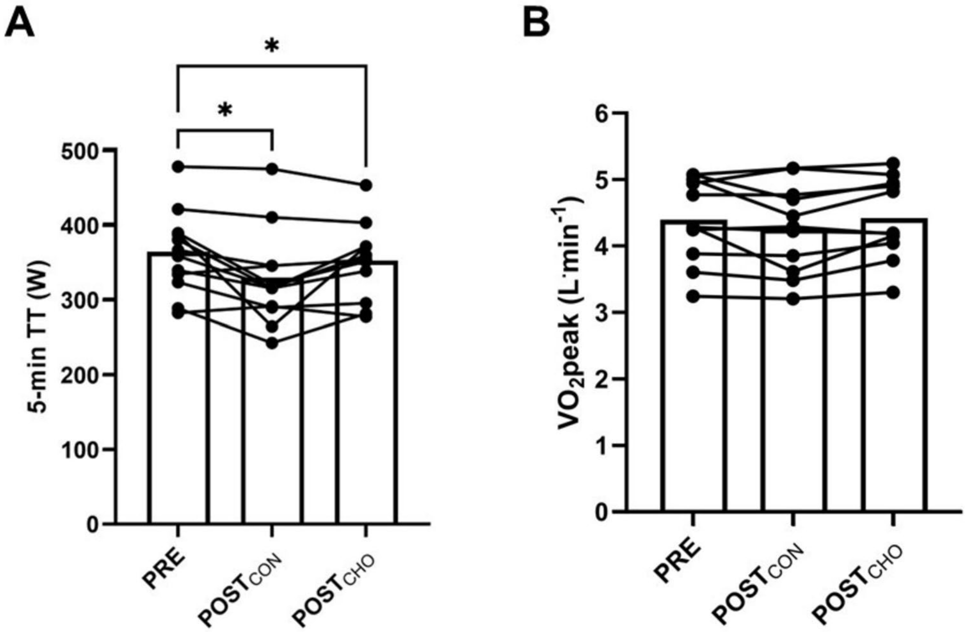

Fig. 4 **A** 5-min time-trial (TT) performance in the PRE, $POST_{CON}$, and $POST_{CHO}$ trials, **B** Peak rate of oxygen consumption (VO_2peak) in the PRE, $POST_{CON}$, and $POST_{CHO}$ trials. * denotes $P \leq 0.05$

2024; Stevenson et al. 2022), and heavy-to-severe (Clark et al. 2019a, b; Clark et al. 2019a, b; Spragg et al. 2024) intensity transitions. Additionally, these results align with previous work reporting that the decline in power output at the heavy-to-severe intensity transition was mitigated with carbohydrate ingestion during exercise (Clark et al. 2019a, b).

Mechanistically, the effect of carbohydrate ingestion on durability of the moderate-to-heavy intensity transition was largely explained by effects on metabolic power, rather than energetic efficiency (Fig. 3B). Metabolic power refers to the rate of whole-body metabolic energy expenditure that is achieved at the moderate-to-heavy intensity transition. The mechanisms for this effect are not clear. Whole-body carbohydrate oxidation rates were ~ 11% greater at the 150-min timepoint during the prolonged phase in $\mathrm{POST_{CHO}}$ compared to $\mathrm{POST_{CON}}$ (Fig. 2A). This modest increase in total carbohydrate oxidation with carbohydrate ingestion is in line with the predictions of a recent meta-regression (Rothschild et al. 2022). Carbohydrate ingestion during prolonged exercise decreases endogenous carbohydrate oxidation rates due to oxidation of the ingested carbohydrate (Gonzalez et al. 2015; Jentjens et al. 2004a, b; Jentjens et al. 2004a, b; Jentjens & Jeukendrup 2005; Wallis et al. 2005). The reduction in endogenous carbohydrate oxidation with carbohydrate ingestion reduces liver glucose output and spares liver glycogen stores (Bosch et al. 1994; Gonzalez et al. 2015; Jeukendrup et al. 1999; McConell et al. 1994). It is therefore possible that the positive effect of carbohydrate ingestion on metabolic power and therefore durability of the moderate-to-heavy intensity transition was mediated by improved regulation of blood glucose concentration due to liver glycogen-sparing (Hargreaves & Spriet 2020; Murray & Rosenbloom 2018). In support, blood glucose concentration was maintained in $\mathrm{POST_{CHO}}$, but declined over time in $\mathrm{POST_{CON}}$ (Fig. 2E). Previous studies have reported that hypoglycemia attenuates central nervous system activation and induces central fatigue. However, this effect is mitigated with carbohydrate ingestion (Elghobashy et al. 2024; Glace et al. 2019; Nybo 2003). Hypoglycaemia-induced central fatigue and consequent impaired motor unit recruitment patterns may have impaired the coordination of muscle contractile function and/or required greater recruitment of type II muscle fibres to produce a given power output, and thus lowered metabolic power and the power output at which participants transitioned from a moderate- to heavy-intensity response. However, it should be noted that a profound hypoglycemia was not observed in $\mathrm{POST_{CON}}$, and the implications of hypoglycemia on submaximal exercise parameters, metabolic power, and energetic efficiency are not clear. As 60 $\mathrm{g{\cdot}h^{-1}}$ of carbohydrate was sufficient to maintain blood glucose concentration (Fig. 2E), future research could investigate the effect of lower rates of carbohydrate ingestion on blood glucose concentrations and durability.

It is also plausible that the greater reduction in metabolic power at $\mathrm{VT_1}$ seen in $\mathrm{POST_{CON}}$ might be related to reduced fiber excitability due to greater depletion of intra- and/or intermyofibrillar glycogen stores. Recent work suggests glycogen-depleted fibres become unexcitable, particularly when exposed to high $\mathrm{K^+}$ concentrations (Cairns & Renaud 2023). An elevated interstitial $\mathrm{K^+}$ concentration may occur due to impaired $\mathrm{Na^+}$, $\mathrm{K^+}$ ATPase function, due to intramyofibrillar glycogen depletion (Nielsen et al. 2022). If carbohydrate ingestion delayed glycogen depletion-induced inactivation in type I fibres, which have more oxidative power and are preferentially activated and therefore glycogen-depleted during prolonged exercise (Nielsen et al. 2024), power output at the moderate-to-heavy-intensity transition may be better preserved. However, most studies suggest that carbohydrate ingestion during prolonged exercise does not preserve muscle glycogen stores (Coyle et al. 1986; Fielding et al. 1985; Flynn et al. 1987; Gonzalez et al. 2015; Hargreaves & Briggs 1988; Jeukendrup et al. 1999; Noakes et al. 1988). However, to our knowledge, no studies have determined the impact of carbohydrate ingestion during prolonged exercise on intramyofibrillar glycogen depletion, which may have specific implications for maintenance of muscle contractile function during prolonged exercise (Nielsen et al. 2022). Therefore, we suggest that future studies investigating the mechanisms by which carbohydrate ingestion during exercise promotes durability of the moderate-to-heavy intensity transition should measure muscle glycogen depletion in specific subcellular depots and individual fibres, and assess effects on central fatigue and motor unit recruitment.

Second, in contrast to our hypothesis, carbohydrate ingestion in $\mathrm{POST_{CHO}}$ did not significantly mitigate the decline in severe-intensity time-trial performance seen in $\mathrm{POST_{CON}}$ (Fig. 4A). Specifically, 5-min time-trial performance was reduced by ~ 4% in $\mathrm{POST_{CHO}}$ and ~ 10% in $\mathrm{POST_{CON}}$, but the difference between $\mathrm{POST_{CHO}}$ and $\mathrm{POST_{CON}}$ was not significant (Fig. 4A). Previous research using a very similar experimental design reported a strong, positive association between durability of the moderate-to-heavy-intensity transition and durability of 5-min time-trial performance (Hamilton et al. 2024). It was hypothesised that this effect was related to having spent less time in the heavy-intensity domain during the prolonged phase (Hamilton et al. 2024). Carbohydrate ingestion did improve durability of the moderate-to-heavy-intensity transition (Fig. 3A), so it is therefore surprising that 5-min time-trial performance was not improved in $\mathrm{POST_{CHO}}$ vs. $\mathrm{POST_{CON}}$ (Fig. 4A). It is possible that the lack of statistical difference in 5-min time-trial performance between $\mathrm{POST_{CHO}}$ and $\mathrm{POST_{CON}}$ was due to the relatively small sample size, although at $n = 11$ with correlation among repeated measures of 0.97 (data not shown), we had 80% statistical power to detect a significant effect with $f > 0.101$. The lack of statistical effect may also have been due to heterogeneity in the level of performance decrement associated with the prolonged exercise protocol (range, + 11 W to -124 W from PRE to POST). As some participants saw very little effect of prolonged exercise on 5-min time-trial performance in $\mathrm{POST_{CON}}$ (Fig. 4A), the prolonged exercise protocol may not have sufficiently demanding to create the conditions for carbohydrate ingestion to improve performance. As blood glucose concentrations were maintained in $\mathrm{POST_{CHO}}$ (Fig. 2E), and given the lack of evidence for a muscle glycogen-sparing effect of carbohydrate ingestion or dose–response relationship between carbohydrate ingestion and performance (Baur et al. 2014; King et al. 2018, 2019; Smith et al. 2010, 2013), it is unlikely that carbohydrate ingestion at a higher rate would have yielded greater performance outcomes. Therefore, it may be necessary to reexamine this hypothesis in a study with a larger sample size and/or a more homogenous cohort undertaking a more prolonged exercise protocol that induces greater fatigue in the control condition.

These data add to our understanding of durability, and have implications for training load monitoring, within-session intensity regulation, and training programming. These data show that carbohydrate ingestion during prolonged exercise allows athletes to maintain a higher power output in relation to their intensity domain transitions. However, inter-individual variability in the effect of prolonged exercise on power output at the moderate-to-heavy intensity transition, and effects of nutrition status, suggests generalised estimates cannot be used when working with individual athletes to account for durability in training programming and quantification of training load. In this study, the effect of the prolonged exercise protocol on power output at the moderate-to-heavy intensity transition ranged from -32 W (-13%) to -3 W (-2%) in $\mathrm{POST}_{\mathrm{CON}}$ and -11 W (-6%) to -3 W (-2%) in $\mathrm{POST}_{\mathrm{CHO}}$ (Fig. 3A). Therefore, future studies should look to develop tools for monitoring power output at the intensity domain transitions in real time that are sensitive to the variable effects of both prolonged exercise carbohydrate ingestion. Development of these tools would allow the intensity of training to be regulated in real time in accordance with dynamic intensity domain transitions, and allow more accurate, physiologically based quantification of training load.

In conclusion, these data demonstrate that carbohydrate ingestion during prolonged exercise partially mitigates the decline in power output at the moderate-to-heavy intensity transition. The positive effect of carbohydrate ingestion on durability of the moderate-to-heavy intensity transition occurred with better maintenance of blood glucose concentrations, and greater preservation of metabolic power at the moderate-to-heavy intensity transition. However, in contrast to our hypothesis, carbohydrate ingestion during prolonged exercise did not mitigate the decline in 5-min time-trial performance. This may have been due to the large variability in responses between-participants as prolonged exercise had little effect on 5-min time-trial performance in some participants, and this may have masked positive effects of carbohydrate ingestion. Our data further highlight the importance of monitoring durability at an individual level due to the large variability between-athletes and the additional complexity that nutritional interventions add to durability responses. Development of methods that accurately quantify power output at physiological thresholds in real time and are sensitive to the various factors, such as nutrition, that impact durability, would also be a significant practical advancement of this research and have direct implications for training load monitoring, training intensity regulation, and predicting performance. Future studies could also explore the effect of carbohydrate ingestion on durability of the moderate-to-heavy intensity transition with measurements of muscle and liver glycogen, neural drive, and muscle excitability.

Authors' contributions H.D.R., C.Z., D.J.P., and E.M. conceived and designed the research. H.D.R. and T.C. undertook the experiments. H.D.R. and E.M. completed data analyses. H.D.R., C.Z., D.J.P., and E.M. drafted the manuscript. All authors revised the manuscript.

Funding None.

Data availability Data are available from the corresponding author upon reasonable request.

Code availability None used.

Declarations

Conflict of interest The authors declare no competing interests associated with this manuscript.

Ethical approval The Auckland University of Technology Ethics Committee approved all procedures in the human studies (23/197).

Consent to participate All participants provided written informed consent.

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